Surgical Treatment of Tricuspid Insufficiency *

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SINCE the advent of open heart surgery, it has become possible to correct most congenital and acquired cardiac lesions. In the majority of patients with mitral valve insufficiency, including those with ruptured chordae tendineae, a good repair of the valve can be obtained as described by Kay, Egerton and Zubiate,[1] Kay, Magidson and Meihaus [2] and Kay and Egerton.[3] Replacement of the mitral valve should be reserved only for the patient in whom a satisfactory valve repair cannot be accomplished. It is only in the minority of patients in whom mitral annuloplasty is not possible that a mitral valve prosthesis should be used in the correction of mitral insufficiency.

Our patients who have had mitral valve annuloplasty for mitral insufficiency or combined stenosis and insufficiency were studied to determine the cause of the 14 per cent mortality (13 of 91 patients). The majority of deaths were in patients with marked cardiomegaly and secondary tricuspid insufficiency. Preoperatively these patients had the classical signs of right-side heart failure and tricuspid insufficiency. The diagnosis of tricuspid insufficiency was substantiated at operation by a large right atrium, a systolic thrill on the right atrial wall and a regurgitant jet in the right atrium. Following operation the patients were awake and rational but blood pressures were low and frequently difficult to maintain at a satisfactory level; there was cyanosis of the lips and extremities and coldness of peripheral tissues and death occurred in several days. This syndrome was associated with a low cardiac output which, in part, was due to a poor myocardium but also aggrevated by large amounts of blood regurgitating through the insufficient tricuspid valve. If these patients had survived the postoperative period the tricuspid annulus undoubtedly would have decreased in size as the right ventricle became smaller during the ensuing months. It appeared necessary to correct the tricuspid insufficiency in order for these very ill patients to survive. For this reason, we instituted a program to achieve tricuspid competency at the same time that the mitral valve was repaired.

Technic of Repair

The tricuspid valve *in situs* is composed of a large posterior leaflet, a somewhat smaller anterior superior leaflet and an even smaller anterior inferior leaflet. Figure-of-eight sutures are placed in the annulus beginning at the commissure between the posterior and anterior inferior leaflet and extending medially toward the commissure between the two anterior leaflets. These sutures obliterate most, if not all, of

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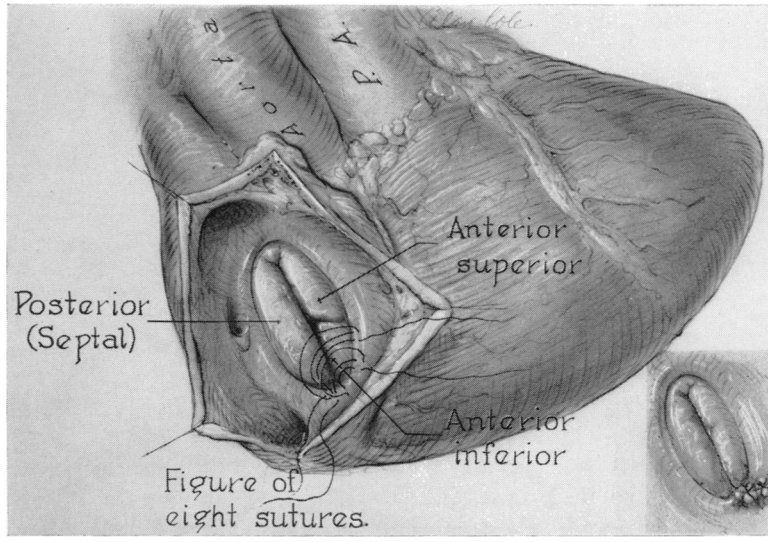

FIG. 1. Method of correcting tricuspid insufficiency. Figure-of-eight sutures of 0 silk are taken in the annulus of the anterior inferior leaflet beginning at the juncture of the posterior and anterior inferior leaflet. Annulus of the posterior leaflet is preserved.

the annulus of the anterior inferior leaflet. Usually three or four sutures of 1–0 silk are required to decrease the size of the annulus from an initial four or five finger breadths to two and one half finger breadths. The sutures are pulled taut but not tied until the surgeon is satisfied with the size of the annulus. After tying the sutures it is difficult to alter the valve configuration. For this reason the repair is maintained by pulling the sutures snug before tying. Basically a large tri-leaflet valve is converted into a smaller mitral like valve (Fig. 1).

Case Reports

Case 1. A 34-year-old woman had rheumatic fever at the ages of 7, 11 and 13 years. A murmur was first heard at the age of seven but she was asymptomatic until the age of 20 when she began to notice dyspnea on exertion. Four years later, following the birth of a child, she developed congestive heart failure and was treated with Digitoxin, diuretics and a low salt diet. Despite this regimen she gradually became worse and for the past 3 years, particularly during the last year, had noticed pronounced symptoms of cardiac failure. At the time of admission she could not do housework and was very dyspneic with minimal exertion. She would have to rest while making her bed and had five-pillow orthopnea with paroxysmal nocturnal dyspnea. Leg edema had been present for months. Diuretics had been discontinued 2 years previously because of jaundice. She had marked abdominal fullness for the previous year.

Physical examination revealed a very sick woman who became dyspneic on talking. Grade II cyanosis of nails and lips was present. There was marked edema of both lower extremities up to the knees, sacral edema and ascites. Marked distention of the neck veins with pulsating V waves were present. A positive hepatojugular reflux was noted. Her liver pulsated and was palpable five finger breadths below the costal margin. Blood pressure in the right arm recumbent was 120/80. Atrial fibrillation was 76. There was a Grade V left ventricular thrust and an apical systolic thrill. A Grade III right ventricular heave was also present. There was a Grade V apical systolic murmur referred to the left axilla and back. A Grade IV to V apical diastolic murmur also radiated to the axilla. An opening snap was present. A Grade III tricuspid systolic murmur was referred to the aortic area but not heard in the neck. There was a right pleural effusion extending half way up the chest. Heart catheterization was not performed.

Prior to operation the patient was treated in the hospital for 2 months for nearly intractable heart failure. She remained at strict bedrest except for use of the lavatory. She was given a 500 mg. salt free diet, diuretics and Digitoxin. Just prior to operation there was no longer leg or sacral edema. The neck veins were much less prominent. Her liver no longer pulsated but was still palpable three finger breadths below the costal margin.

At operation, grading from I to VI, the right atrium was Grade III enlarged, the left atrium Grade VI enlarged, the left ventricle Grade III enlarged and the right ventricle Grade II en-

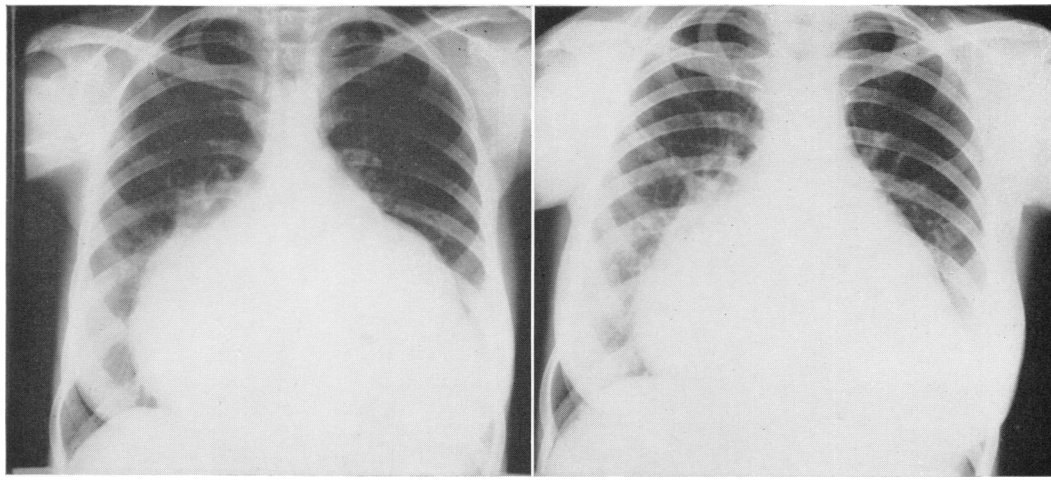

FIG. 2. X-ray before and 6 months after mitral and tricuspid annuloplasty. Despite excellent repair and marked improvement, heart is still enlarged.

larged. Pulmonary artery pressure appeared to be about one third of the systemic pressure. There were Grade II systolic thrills in the left and right atria. The mitral valve was free of calcium and markedly insufficient. The mitral ring easily admitted four fingers. The mural leaflet was quite firm and a 4-mm. plaque of calcium was present near the anterolateral commissure. Using the technic described by Kay, Egerton and Zubiate,[1] four interrupted figure-of-eight sutures of 2–0 silk were placed at the posteromedial commissure in order to narrow only the mural annulus. At the end of the procedure the mitral annulus admitted two fingers. Although the chordae tendineae appeared redundant, it was thought that the repair was adequate. The tricuspid valve also admitted four fingers easily. Tricuspid annuloplasty was performed as described above. The duration of cardiopulmonary bypass was 42 minutes. A tracheostomy was performed and the patient was maintained on an Engstrom Respirator in the postoperative period.

She was discharged home on the 30th postoperative day after a slow but uncomplicated hospital course. Eight months after operation she is doing well and carrying on normal activities (Fig. 2).

Case 2. A 27-year-old woman had rheumatic fever at the age of nine, at which time she was in bed for three months. Following this she had aches and pains frequently and was out of school much of the time. She was hospitalized for 3 years, until the age of 13, because of rheumatic fever. Since then she had been hospitalized many times. At the age of 15 she developed congestive failure and was given digitalis. She was taking digitalis and a low salt diet when seen. There was no orthopnea nor paroxysmal nocturnal dyspnea but she did have difficulty doing housework and noticed dyspnea on climbing one flight of stairs. System review was essentially negative except for a previous tubal ligation.

On physical examination the blood pressure in her right arm recumbent was 160/70. Atrial fibrillation was present and there was a pronounced right ventricular lift as well as a left apical thrust at the anterior axillary line at the left fifth intercostal space. There was a Grade IV apical systolic murmur, an opening snap and a Grade II to III apical diastolic murmur. There was a Grade IV systolic murmur at the tricuspid area and a Grade I diastolic murmur noted to the left of the sternum. Her liver was palpable and pulsating four finger breadths below the costal margin. There was a positive hepatojugular reflux and pronounced distention of neck veins with marked V waves.

Right heart catheterization was performed in 1955, and this revealed a pressure of 36/17 in the pulmonary artery, 42/10 in the right ventricle and 11/8 in the right atrium. Catheterization was not repeated.

Operation was performed on June 25, 1963, at which time she was found to have mitral stenosis and insufficiency with secondary tricuspid insufficiency. The mitral valve was thick, with both commissures fused, and admitted one and one-half fingers. The valve was repaired by opening the anterolateral and posteromedial commissures and performing a posteromedial annuloplasty. The tricuspid annulus was very large and admitted four and one-half to five fingers. Four interrupted figure-

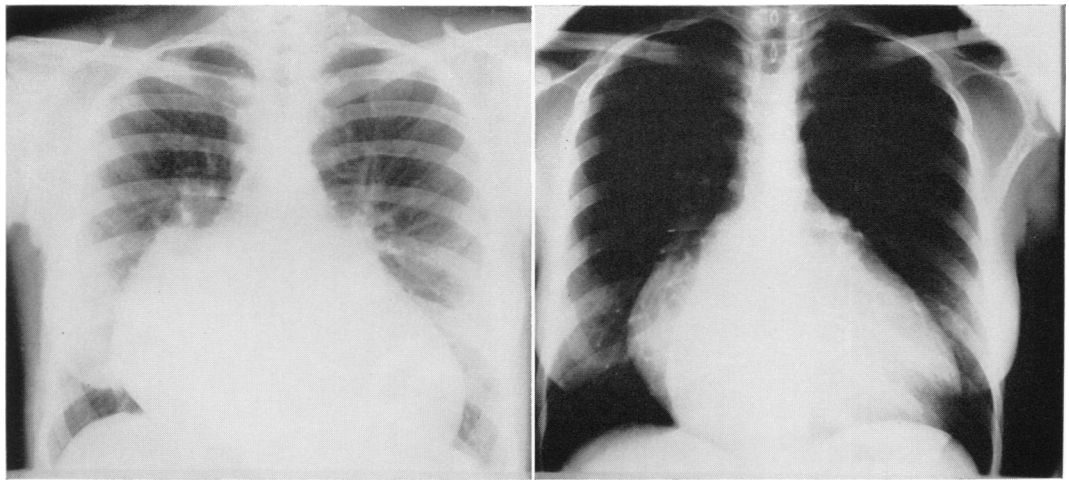

FIG. 3. Pronounced cardiomegaly prior to operation with reduction in size 10 months following mitral valve replacement and tricuspid annuloplasty.

of-eight sutures of 1–0 silk were used to narrow the tricuspid valve to a two finger-breadth opening. At the end of cardiopulmonary bypass, a Grade I to II systolic thrill was felt in the left atrium. It was thought that this patient would need perfectly functioning mitral and tricuspid valves in order to survive and, for this reason, cardiopulmonary bypass was reinstituted and the mitral valve was replaced with a No. 3 Starr-Edwards ball valve.

Postoperatively she developed a hemolytic *Staphylococcus aureus* coagulase positive endocarditis which was treated successfully with penicillin, 60,000,000 units daily for 6 weeks, Prostaphlin, 8 Gm. daily for 6 weeks, Streptomycin, two Gm. daily for 10 days, Chloromycetin, 2 Gm. daily for 10 days and Erythromycin, 1 Gm. daily for 8 days. Eleven months following operation and she is doing well (Fig. 3).

Results

Twenty patients were operated upon between March, 1960 and February, 1964 for rheumatic mitral valve disease and secondary tricuspid insufficiency. This represents 8% of the operations performed for mitral valve disease. There were 12 women and eight men. The average age was 38.5 years; the youngest patient was 17 and the oldest 56 years of age. Three patients had pure mitral stenosis, one had pure mitral insufficiency and 16 had varying degrees of mitral stenosis and insufficiency. In 12 patients it was necessary to replace the mitral valve with a prosthesis; five of these 12 died. In the remaining eight patients, mitral commissurotomy alone or with annuloplasty was performed with one death. There were, therefore, six deaths in this group of 20 patients. The 14 surviving patients have done well and in one there had been not only mitral valve disease but also a minimal degree of aortic stenosis. Valvuloplasty was performed for both the aortic and mitral lesions in this patient. The average age of the patients who died was 44.5 years and this was higher than the average age of 36 years of the surviving group.

The most common clinical sign was shortness of breath with exertion and this was present in all patients. Twelve had signs of right-sided heart failure at the time of admission to the hospital. Only five had specific signs of tricuspid insufficiency, relieved by distended neck veins with large V waves, pulsating liver and a positive hepatojugular reflux. One patient had a positive Rivero-Carvallo sign as manifested by the increase of the systolic murmur in the tricuspid area during deep inspiration. Seventeen of the 20 patients had atrial fibrillation at the time of operation.

Discussion

Twelve patients had signs of heart failure at the time of hospital admission. The remaining eight did not have obvious cardiac failure because of the stringent medical therapy which was instituted with strict bedrest at home for several months prior to entering the hospital for operative treatment. Even after 2 to 3 months of strict bedrest and proper care some patients still had pronounced tricuspid insufficiency at the time of operation.

In 1962, Salazar and Levine [4] stated that tricuspid insufficiency is never isolated but that mitral valve disease is integral to this disorder. This finding has been confirmed in our experience and noted in the previous publication by Zubiate and Kay.[5] The patient with mitral and tricuspid valve disease also may have associated aortic valve disease, usually predominantly stenosis rather than insufficiency. We have not seen a patient with aortic and tricuspid involvement without concomitant mitral valve disease. With rheumatic involvement of the tricuspid valve, tricuspid stenosis appears to be the rule. There may be superimposed tricuspid insufficiency with the tricuspid stenosis but this seems to be present only in the patient with atrial fibrillation.

Salazar and Levine [4] suggested surgical correction for tricuspid insufficiency of organic origin. Our concern has been limited to tricuspid insufficiency secondary to mitral valve disease since we believe that pure tricuspid insufficiency is always functional and not due to rheumatic involvement of the tricuspid valve. If the tricuspid valve is so involved the valve should show evidence of tricuspid fusion.

Tricuspid insufficiency may be associated with pulmonary hypertension. Usually the patient with tricuspid insufficiency associated with predominant mitral stenosis develops tricuspid insufficiency following severe pulmonary hypertension, after which there may be myocardial failure and subsequent tricuspid insufficiency. Pulmonary hypertension may not be excessive at the time of right heart catheterization because pulmonary artery pressure may fall with the onset of tricuspid insufficiency. Patients with predominant mitral insufficiency usually develop tricuspid insufficiency in the absence of pulmonary hypertension; myocardial failure, unrelated to pulmonary hypertension, is the predisposing factor.

A group of patients with tricuspid insufficiency was not included in this report. These patients had the classical findings of tricuspid insufficiency when first seen but the insufficiency disappeared on a proper medical regimen and was not a factor at operation. Tricuspid insufficiency has occurred at the conclusion of bypass after mitral valve surgery but usually disappeared before the chest was closed—sufficiently mild not to require tricuspid annuloplasty. This transient tricuspid insufficiency is probably due to myocardial failure secondary to cardiac manipulation and bypass, evidenced by its disappearance with the use of adrenergic drugs, digitalis and supportive measures in the immediate postbypass period.

If there is moderate-to-pronounced tricuspid insufficiency in the postoperative period the right ventricle may not sustain an adequate cardiac output. If the tricuspid valve were not repaired and, despite a low cardiac output, the patient were to survive the postoperative period, the right ventricle would become smaller in succeeding months in the presence of a functioning mitral valve, as would the tricuspid annulus, and the tricuspid valve would be competent. However, chances of survival are lessened in the presence of significant tricuspid insufficiency.

Summary

Twenty patients with functional tricuspid insufficiency secondary to mitral valve disease have been operated upon. In all in-

stances the tricuspid insufficiency was sufficiently significant to require repair. The mitral valve was repaired in eight patients and replaced with a ball valve prosthesis in 12. One patient also had an aortic commissurotomy for aortic stenosis. There were six deaths in these 20 patients—five in the 12 patients with mitral valve replacement.

It is concluded that tricuspid annuloplasty is an important adjunct to mitral valve surgery in increasing survival in critically ill patients. There would appear to be little, if any, place for a tricuspid prosthesis in the treatment of functional tricuspid insufficiency.

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